Correspondence

The Editorial Board will be pleased to receive and consider for publication correspondence containing information of interest to physicians or commenting on issues of the day. Letters ordinarily should not exceed 600 words, and must be typewritten, double-spaced and submitted in duplicate (the original typescript and one copy). Authors will be given an opportunity to review any substantial editing or abridgement before publication.

Treatment of Rattlesnake Bites

TO THE EDITOR: In the correspondence on rattlesnake bites in the January 1984 issue, Dr A. M. Butner appears to imply that the venom of the northern Pacific rattlesnake, *Crotalus viridis oreganus (Cvo),* is substantially different and less toxic than that of the southern Pacific rattlesnake, *Crotalus viridis helleri (Cvh).*[1] Unfortunately, there are no experimental or clinical data to support such a contention. The LD_{50}s for the venom of the two subspecies are identical, as are their separations by electrophoresis, their patterns on isoelectric focusing, ELISA (enzyme-linked immunosorbent assay) findings and all physiopharmacological measurements to which the two venoms have been subjected. There is as much difference in the composition of the venoms of individual snakes of one subspecies as there is between the venoms of the two subspecies. In no way can the two subspecies be divided on the basis of their venom. In fact, they are divided only by an imaginary geographical line running from Lebec-Shandon–Cape San Marin in Kern and San Luis Obispo counties, California, and by indifferent terminal rings on the tail. Having treated well over 300 patients bitten by these two subspecies, I feel I would be at a loss to distinguish any clinical differences in either the symptoms and signs of envenomation, or the severity of poisoning following the bites of the two subspecies.

I think Dr Butner has just been lucky (which has been my experience for the same area) in that the patients he has reported on have not been bitten by large members of the subspecies. The number of bites by *Cvo* is certainly far less than that for the southern subspecies. However, I am sure that Dr Butner is aware that there have been deaths due to the bites of *Cvo.* Over the past 30 years I have consulted on numerous cases in which severe tissue changes ensued following the bite of this subspecies, principally consultations from northern California and Oregon.

I agree with Dr Wingert[2] that the grading system used by Dr Butner is not applicable to bites by the western species of rattlesnake, and is slowly losing favor elsewhere. I am rather surprised that Dr Butner states that the grading system which Dr Wingert refers to "does not lend itself to a sensitive adjustment of antivenin dosage as the toxic state progresses," for that is the *very* reason that that system was developed. Dr Butner's statement is almost the opposite of the argument I presented in my book[3] for *not* using the system he suggests.

> The grading methods of 0 or 1 through 4 or 5, which were suggested and used many years ago,[4,5] are precarious . . . they are usually based on a few selected symptoms or signs, and these are often measured at 12 hours following the bite. The physician should base his diagnosis . . . on all clinical manifestations, including changes in the blood cells and chemistry, changes in motor and sensory function. . . .[3]

I feel the "numbers system" is useful only to those who know what their own numbers mean. My experiences in the courts would seem to indicate that there is no common definition or agreement on these numbers for grading.

Finally, it is to be hoped that clinicians will never base their treatment on the "virulence of local reptiles." We are treating patients, not reptiles, and it is the patient's symptoms and signs and the severity of the poisoning to which our attention should be directed.

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Ergonovine-Induced Chest Pain Not Due to Coronary or Esophageal Spasm

TO THE EDITOR: We would like to comment on the informative paper by Lieberman and co-workers, "Ergonovine-Provoked Esophageal Spasm During Coronary Angiography," in the March issue.[1]

We concur with the concept of performing simultaneous coronary angiography and esophageal manometry during provocative ergonovine maleate testing. We consider an important observation to be the chest pain induced by ergonovine in patient 9 in the current study, accompanied by neither angiographic nor manometric changes at the time of pain. We have had similar findings in three patients, although two of them had minor underlying manometric abnormalities not exhibited at the time of chest pain following ergonovine. Cardiac metabolic studies were not carried out but there was a rise in systemic arterial pressures and a